Susceptibility-Weighted MRI and Microbleeds in Mild Traumatic Brain Injury: Prediction of Posttraumatic Complaints?

Gerard Hageman^a Jurrit Hof^b Jik Nihom^a

^aDepartment of Neurology, Medical Spectrum Twente, Hospital Enschede, Enschede, The Netherlands;

^bDepartment of Radiology, Medical Spectrum Twente, Hospital Enschede, Enschede, The Netherlands

Keywords

Mild traumatic brain injury · Posttraumatic complaints · Susceptibility-weighted MRI · Microbleeds · Traumatic axonal injury

Abstract

Background: Only in 7–15% of patients with mild traumatic brain injury (mTBI), traumatic CT-abnormalities are found. Nevertheless, 40% of mTBI patients suffer from posttraumatic complaints not resolving after 6 months. We discuss the ability of susceptibility-weighted imaging (SWI), sensitive for microbleeds, to detect more subtle brain abnormalities.

Summary: After a search on PubMed, we selected 15 studies on SWI in adult mTBI patients; 11 studies on 3T MRI, and 4 studies on 1.5T MRI. All 1.5T studies showed that, compared to T2, gradient echo, diffusion-weighted imaging, or fluid-attenuated inversion recovery sequences, SWI is more sensitive for microbleeds. Only two 1.5T studies described the association between SWI findings and outcome. In 3 of the 4 studies, no control group was present. The mean number of microbleeds varied from 3.2 to 6.4 per patient. In the 3T studies, the percentage of patients with traumatic microbleeds varied from 5.7 to 28.8%, compared to 0–13.3% in normal

controls. Microbleeds were particularly located subcortical or juxtacortical. The number of microbleeds in mTBI varied from 1 to 10 per patient. mTBI patients with microbleeds appeared to have higher symptom severity at 12 months and perform worse on tests of psychomotor speed and speed of information processing after 3 and 12 months, compared to mTBI patients without microbleeds. **Key Messages:** There is some evidence that traumatic microbleeds predict cognitive outcome and persistent posttraumatic complaints in patients with mTBI.

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Introduction

The large majority of traumatic brain injuries (70–90%) can be classified as mild traumatic brain injury (mTBI), which is indicated by a Glasgow Coma Scale (GCS) score between 13 and 15 on admission to the emergency department, loss of consciousness <30 min, and posttraumatic amnesia <24 h. In more than 80% of patients with mTBI in Dutch emergency departments, a non-contrast head CT is performed [1, 2]. Intracranial traumatic findings are found in 7–15% of those CTs. Less

than 1% of mTBI patients require neurosurgery, most often due to sub- or extradural hematomas [2, 3]. The majority of patients report complete symptom resolution after 3–6 months following mTBI. However, a subset of patients report post-concussion symptoms, notwithstanding an initial normal head CT. These symptoms can be defined as somatic, cognitive, or emotional, and sometimes symptoms may persist for months or even years [4]. MRI is seldom performed as an evaluation tool for acute mTBI, mainly because of logistic and financial reasons. Despite the lack of evidence, the current practice is to perform MRI in patients with mTBI with persistent posttraumatic cognitive complaints after 3–6 months, after a normal CT [5]. Pulse sequences used for MR imaging in TBI include conventional T1-weighted, T2-weighted (T2W), fluid-attenuated inversion recovery (FLAIR), T2-gradient echo (GRE), and diffusion-weighted imaging. GRE sequences have long been used as the method of choice to detect blood products and calcifications. This sequence shows local susceptibility effects resulting from the small field inhomogeneities caused by microbleeds. Still head CT and this conventional MRI protocol are not sensitive enough for the detection of small hemorrhagic foci.

Susceptibility-weighted imaging (SWI) is a recently added and promising imaging tool to detect microbleeds [6]. Although SWI sequences do take more time (5–7 min) to acquire than T2-GREs, the sensitivity of SWI by far exceeds a T2-GRE technique. Pediatric TBI studies have found six times as many lesions with SWI as with GRE. Compared to CT scanning and conventional MRI in 30% of the time, additional lesions were found using SWI [7, 8]. SWI is especially sensitive to hemorrhagic lesions, not only supratentorially but also in the posterior fossa [9, 10]. Furthermore, SWI displays the boundaries and extent of the microbleeds more clearly than traditional GRE sequences [11].

The challenge in mTBI is to predict which patients are likely to develop chronic symptoms. It appears that patients with traumatic microbleeds in the acute phase of mTBI show worse performance in cognitive function shortly after trauma and at long-term follow-up, as well as higher post-concussion symptom severity [12]. It is uncertain whether the number of microbleeds provides prognostic information, although there is some evidence that higher lesion volume is associated with poorer memory and impairment of processing speed [13]. In addition, the localization of microbleeds may be important. Microhemorrhages in the temporal cortical area after mTBI may be associated with an unfavorable outcome [14, 15]. Traumatic microbleeds remain visible for up to 5 years,

even though their numbers may slightly reduce over time [16, 17].

In this narrative review of the literature, the following questions are addressed: (1) What is the prevalence, localization, and number of microbleeds in mTBI, detected by SWI, (2) Is the presence of microbleeds a predictor for persistent posttraumatic complaints or an unfavorable long-term outcome in mTBI, and (3) Is it possible to provide a radiological analog for posttraumatic complaints based on the presence, quantity, or location of traumatic microbleeds?

SWI

SWI is a relatively new MRI technique, available for clinicians for nearly 20 years. SWI enhances contrast comparing varying tissue types to surrounding structures by utilizing the inherent variation in magnetic susceptibility [10]. Iron at sites of previous hemorrhage is associated with strong susceptibility variations. This creates local distortions in the magnetic field and differences in the phase of the acquired MRI data [10]. SWI combines the conventional magnitude image with a filtered phase image, which reduces artifacts and background field inhomogeneities from the original phase image. The increased sensitivity and phase mapping of SWI make it possible to distinguish paramagnetic (hemorrhage/iron) from diamagnetic (calcifications) substances; hemorrhages are highlighted [18]. Nevertheless, some structures or lesions exhibit susceptibility effects that can mimic microbleeds, such as iron deposits in the basal ganglia and artifacts from bone-air interfaces [19]. Despite this “overdetection” of hemorrhagic lesions, SWI now is the golden standard to detect small hemorrhagic foci (diameter <10 mm) and has become a standard addition to routine TBI-MRI protocols [11, 20, 21].

Field Strength

Studies have reported conflicting results regarding the association of SWI findings with clinical and outcome variables. Using the GCS, in some studies, SWI was a non-superior outcome predictor compared to T2W and FLAIR images [22]. In most studies, however, microbleeds on SWI predict an almost double risk of disability at 1 and 3 months after the injury [23]. This detection of traumatic cerebral microbleeds is heavily influenced by field strength (1.5 T vs. 3 T) and the echo times involved [20]. Thus stud-

Fig. 1. Search strategy.

ies using field strengths of 1.5 T may underestimate the presence of hemorrhagic lesions [24, 25]. Studies using 3T SWI are able to show much smaller microbleeds, especially when longer echo times are used. Ultra-high field 7T MRIs detect even smaller and more microbleeds, however at the cost of increased artifacts [24, 26, 27]. In this paper, we will only review 3T and 1.5T SWI studies, as these field strengths are available in everyday practice.

Methods

A literature search on PubMed for studies from 2005 to September 2021 evaluating SWI as a predictive factor of posttraumatic complaints or unfavorable cognitive outcome in adult populations with mTBI was performed (Fig. 1). We excluded SWI studies with a focus on iron deposition in chronic mTBI.

Cerebral Microbleeds

Cerebral microbleeds are defined as hemorrhagic, T2-, and susceptibility-weighted hypointense lesions less than 10 mm in diameter without connection to the brain surface or ventricular system, and at least half surrounded by brain parenchyma [7]. Additional recommended criteria are black and blooming appearance on T2W MR imaging, round or ovoid, devoid of signal hyperintensity on T1W or T2W sequences [28]. Cerebral microbleeds resulting from TBI are distinct from those due to cerebral amyloid angiopathy or hypertensive angiopathy [29, 30]. The key imaging features of cerebral amyloid angiopathy are lobar, often in the parietal and occipital lobes, cortical, or subcortical microbleeds. In contrast, hypertensive angiopathy is characterized by microhemorrhages predominantly infratentorial and in the basal ganglia, to a less degree in the thalami, and periventricular white matter.

Table 1. 3T SWI studies of patients with mTBI

Authors	Interval between mTBI and SWI	Study design, mTBI patients, <i>n</i> , mean age	controls, <i>n</i> , mean age	SWI findings, relation to symptoms, neuropsychological findings
Toth et al. [39]	T1: <3 days T2: after 1 month	Longitudinal case-control study 14 patients; 9 M and 5 F, GCS 15, LOC <1 min, head CT and conventional MRI: normal; minimal head trauma; 34.3 years (20–72)	14 healthy controls, 35 years (20–71)	No microbleeds in mTBI or controls, on T1 and T2
Wang et al. [33]	<3 days	Prospective study, comparing SWI findings in patients with and without depression after mTBI 165 patients; group 1: 28 patients with posttraumatic depression <1 year, 39.9 years. Group 2: 137 patients without depression, 44.9 years	Group 1 versus group 2	In 32 of 165 patients (19.4%) microbleeds, especially in the subcortical white matter frontal, temporal, and parietal Group 1: in 20 of 28 patients 1 or more microbleeds (71.4%) Group 2: in 12 of 137 1 or more microbleeds (9%)
Huang et al. [34]	Mean 25 days (8–55)	Prospective case-control study, SWI and digit span test 111 patients; 43 M 68 F, normal head CT and conventional MRI, 37.1 years (21–84)	111 healthy controls 39.6 years (18–61)	60 microbleeds in 26 of 111 patients (28.8%), subcortical white matter 15 microbleeds in 12 controls (13.3%), mainly basal ganglia, and thalamus. mTBI patients with microbleeds perform worse on digit span (short term memory) task than patients without microbleeds
Tate et al. [38]	Mean 308 days (3–24 months)	Prospective cohort study, FLAIR and SWI 77 patients; 72 M 5 F, normal CT, persistent posttraumatic complaints 32.8 years (18–55)	58 orthopedic controls and 17 patients with posttraumatic stress syndrome	Microbleeds in 22% of mTBI (gray/white matter junction, corpus callosum, posterior fossa), in 1% of orthopedic controls and in 0% of PTSS. mTBI patients with FLAIR white matter hyperintensities perform worse on working memory task compared to control groups. With or without microbleeds: no significance
Trifan et al. [37]	Mean 29 months (2–158 months)	Retrospective case-control study, FLAIR and SWI 150 patients with persistent complaints; 88 M and 62 F, 43 years (14–81)	94 healthy controls, 58 years	Microhemorrhages and hemorrhagic contusions in 26 patients (17%) Subgroup analysis mTBI patients <45 years: 11 of 73 (15%) microbleeds; mTBI patients >45 years: 15 of 77 (19.5%) Microbleeds in 3 of 94 controls (3.2%)
Studerus-Germann et al. [10]	T1: <1 week T2: 12 months Neuropsychological testing at 1 week, and after 3 and 12 months	Prospective longitudinal cohort study, post-concussion symptoms, cognitive testing and T1, T2, DTI, and SWI 30 patients; 16 M and 14 F, normal CT, persistent posttraumatic complaints, 35 years (18–55)	20 healthy controls, 43.2 years (19–62)	T1: in 4 of 30 (13.3%) microbleeds, hemorrhagic contusions T2: in 4 of 20 (20%) microbleeds, hemorrhagic contusions In 5 of 20 (25%) controls: microbleeds Cognitive testing at T1: Significant differences in 8 tests (psychomotor speed and speed of information processing) in patients with and without microbleeds; At T2: Significant differences still in 5 tests Higher symptom severity in patients with microbleeds (fatigue, impaired memory and concentration) Number of microbleeds at T1 correlates with higher symptom values at T2
Van der Horn et al. [35]	4 weeks	Prospective multicenter cohort study, questionnaire, SWI, and GRE 54 patients; 36 M and 18 F with normal CT; questionnaire after 2 weeks: 34 patients posttraumatic complaints, 20 patients: no complaints 37 years (19–64)	20 healthy controls 34 years (18–61)	A total of 158 microbleeds (mainly superficial, frontal, temporoparietal, and corpus callosum) in 15 of 54 patients (28%) and in 0% of controls. No statistical differences in SWI abnormalities and patients with or without (early) posttraumatic complaints

Table 1 (continued)

Authors	Interval between mTBI and SWI	Study design, mTBI patients, <i>n</i> , mean age	controls, <i>n</i> , mean age	SWI findings, relation to symptoms, neuropsychological findings
Toth et al. [15]	Mean 2 years, SD 15 months	Retrospective study, head CT, T1, T2, FLAIR, and SWI 13 mTBI patients (GCS 14–15), 4 moderate, 21 severe TBI, 39.7 years (18–83)	20 healthy controls	In 1 of 13 (7.7%) mTBI patients (subcortical) microbleeds, in 1 of 4 (25%) moderate and in 12 of 21 (57%) severe TBI
Einarsen et al. [40]	T1: <72 h T2: At 3 months (91–99 days) (<i>n</i> = 165) T3: At 1 year (364–374 days) (<i>n</i> = 152)	Prospective longitudinal cohort study, T2, FLAIR, SWI, and post-concussive symptom inventory 194 patients; 124 M 70 F, GCS 15: 149; abnormal CT: 13 of 162 patients (8%) At 3 months: 21% of mTBI patients: post-concussive syndrome At 12 months: 15% 27 years	184 mTBI patients (age 24 years) received basic follow-up and telephone interview; and 78 “community controls”: MRI protocol	In 23 of 194 patients traumatic MRI lesions, of which 11 patients (5.7%) with microbleeds. At T2 and T3 microbleeds persisted, although smaller and less uniformly hypointense In 2 of 78 controls (2.6%) microbleeds At 3 months 7 (33%) of patients with and 30 (19%) of patients without traumatic MRI findings met criteria for PCS At 12 months resp. 4 (21%) and 21 (14%) At 12 months 16% of patients with PCS and 11% of patients without PCS had traumatic MRI findings, not significant
Griffin et al. [23]	<48 h	Prospective, multicenter study, DTI, GRE, SWI, FLAIR, and 3D-T1 sequence; GOSE at 30 and 90 days 365 patients with mTBI, 55 moderate, 19 severe TBI, median age 46 years	Patients with and without microbleeds; and patients with GOSE 1–6, compared to GOSE 7–8	Traumatic microbleeds in 27% of mTBI, 47% of moderate and 58% of severe TBI. Location mainly frontal, temporal, and parietal lobe In binary logistic regression analysis, both linear and punctate microbleeds were significant predictors of outcome
Huovinen et al. [36]	3–17 days	Prospective cohort study, post-concussion symptom questionnaire, GOSE, time to RTW 113 patients, 64 M 49 F, mean GCS score 14.7 39.2 years, SD 12.2	Patients with microbleeds compared to patients without microbleeds	22 patients (19%) microbleeds: more often motor vehicle accident, LOC; RTW after 13.5 days, compared to 91 patients without microbleeds: after 7 days Post-concussive symptoms and GOS-E: no significant differences
LOC, loss of consciousness; PCS, post-concussive syndrome; DTI, diffusion tensor imaging; GOSE, Glasgow Outcome Scale Extended; RTW, return to work.				

The prevalence of cerebral microbleeds increases with age from 17.8% in asymptomatic persons aged 60–69 years to 38.3% in those over 80 years [31]. Nontraumatic microbleeds were detected by SWI in 40% of patients presenting at a memory clinic [32]. So distinguishing between traumatic and nontraumatic microbleeds is especially clinically relevant in older victims of mTBI.

Results

In Table 1, we summarize the results from 3T studies, and in Table 2, those from 1.5T studies. We describe 11 3T studies of SWI in mTBI in detail (Table 1). In 5 studies, SWI was performed in the early posttraumatic phase (<1 month) [27, 33–36], in 3 in the late phase [15, 37, 38], and 3 studies were longitudinal [12, 39, 40]. Most studies

were prospective cohort or case-control studies. Trauma severity in mTBI patients varied from minimal head trauma [39], GCS 15 and loss of consciousness <1 min, to GCS 14–15 [15] with normal head CT [34, 38], and GCS 13–14 and abnormal CT in 8% [40]. In patients with minimal head trauma, no traumatic microbleeds were found. In mTBI patients, the percentage of patients with microbleeds varied from 5.7 to 28.8%, compared to 0–13.3% in controls. In 1 small study of 30 mTBI patients and 20 healthy controls, the percentages were not differentiating and remarkably high (25%) in healthy controls [12]. The four largest studies with >100 patients confirmed the percentages as mentioned above [23, 34, 37, 40]. The number of microbleeds in patients after mTBI varied from “1 or more” [33], to 158 microbleeds in 15 patients [35]. Hemorrhagic foci after mTBI may have diameters of 10–20 mm [11]; however, traumatic microbleeds are small,

Table 2. 1.5T studies of SWI in mTBI patients

Authors	Interval between mTBI and SWI	Study design, mTBI patients, <i>n</i> , mean age	Controls, <i>n</i> , mean age	SWI findings, relation to symptoms, and outcome
Park et al. [41]	<1 week	Case-control study; 21 patients, 15 M and 6 F, with normal conventional MRI, 37.2 years	42 headache patients, no trauma	51 microbleeds in 16 of 21 (76%) patients; 44 of 51 microbleeds in frontal and occipital white matter, 7 in basal ganglia, thalamus 9 of 16 patients symptomatic: disorientation, visual field defect, hearing difficulty 16 microbleeds in 10 of 42 (23%) controls, especially in basal ganglia and thalamus Patients with microbleeds: mean GCS 13.9, GOS at 1 year 4.7 Patients with >7 microbleeds: mean GCS 13.1, GOS at 1 year 4.1 Patients without microbleeds: mean GCS 15, GOS at 1 year 5
Akiyama et al. [43]	5–10 days	Case report; 2 patients, 2 M, both with microbleeds, 41 and 59 years	T2W imaging compared to SWI	Total number of microbleeds on T2: 8 Total number of microbleeds on SWI: 40
De Haan et al. [14]	24 weeks	Retrospective cohort study; 127 patients, 74 M 53 F, with persistent posttraumatic complaints >3 months after injury; cognitive complaints 87% 39 years (16–76)	T2 GRE compared to SWI	SWI: 341 microbleeds in 63 of 127 patients (50%), frontal, temporal white matter T2: 179 microbleeds Patients with microbleeds: lower GCS, more frequent LOC, and PTA; unfavorable GOS-E: 67% Patients without microbleeds: unfavorable GOS-E in 40% Worse GOS-E associated with total number and temporal subcortical area localization of microbleeds
Eldeş et al. [42]	6 h	Case series; 45 patients, 23 M and 22 F, with complaints at 6 h after injury (headache, vertigo, nausea, concentration↓; GCS 15: 44 patients GCS 14: 1 patient 47.9 years	Comparing T2, DWI, FLAIR, and SWI	SWI: 45 microbleeds in 7 of 45 patients (15.6%) T2/FLAIR: 5 of 7 abnormal (hyperintense lesions) SWI: greater number of microbleeds and larger in size
DWI, diffusion-weighted imaging; LOC, loss of consciousness.				

punctate, or linear, particularly located sub- or juxtacortical in the frontal, temporal, and parietal lobes [14, 15, 23, 33–35, 38], less frequently in the posterior fossa and corpus callosum [35, 38]. In some studies, the relation between microbleeds and persistent posttraumatic complaints, cognitive performance, or outcome is described. There was no association between microbleeds and early symptoms at 4 weeks [35]; however, mTBI patients with microbleeds showed higher symptom severity at 12 months [12]. Remarkably, in 1 study, microbleeds were correlated with a major depressive episode within 1 year after mTBI [33]. Among 165 patients, 20 of 28 depressive and 12 of 137 non-depressive patients showed microbleeds. In 3 studies, the cognitive performance of mTBI-patients with microbleeds was compared with patients

without microbleeds. There were significant differences in tests of psychomotor speed, speed of information processing after 3 and 12 months, and in short-term memory (Digit Span) after 1 month [34, 40]. In contrast, no significant differences were found on a working memory task, evaluated after an interval of 3–24 months [38] (Table 1).

In Table 2, we describe four 1.5T SWI studies in mTBI in detail [14, 41–43]. In three of these studies, SWI was performed within 1–10 days after trauma [41–43]. SWI was compared to T2, GRE, diffusion-weighted imaging, or FLAIR sequences in 3 studies [14, 42, 43]. In 1 study, the percentage of mTBI patients with microbleeds was very high (76%), but so was the percentage (23%) of microbleeds in controls, suggesting a nontraumatic origin

[41]. In other studies, 15.6–50% of patients had microbleeds. The mean number of microbleeds varied in these 1.5T studies from 3.2 to 6.4 per patient [14, 41, 42]. Only two studies described the association between SWI findings and outcome [14, 41].

SWI studies <1 week after mTBI may be difficult to interpret [44]. Microbleeds are not static, but change in number and volume over time, especially in the acute phase. Imaging time is relevant for optimizing the prognostic utility of SWI. More specific, due to the biophysical properties of microbleeds, they become isointense to the white matter and temporarily less detectable at 24–72 h following injury [45].

Microbleeds are commonly associated with traumatic axonal injury (TAI) [6]. The same external shearing force that leads to TAI causes disruption of adjacent small vessels [23]. The presence of traumatic microbleeds in the brainstem and corpus callosum on SWI images correlates with neuropathological findings of TAI [46]. There are no postmortem imaging and histology studies of mTBI patients, to correlate pathological and radiological findings. Direct comparison of histology of microbleeds with postmortem MR images in other studies only included non-traumatic microbleeds [47, 48].

Discussion

Post-concussion syndrome is a constellation of physical, cognitive, and emotional symptoms [49]. In mTBI, most patients show an amelioration of symptoms over time [50]. However, in a subpopulation after mTBI, symptoms continue to emerge for months subsequent to injury, persisting longer than 1 year, sometimes even without any trend toward improvement [49]. Predictors for 6-month outcome are education, age, emotional distress, and coping [4]. In addition, it may be useful to examine the contributing role of the mechanism of injury or pre-injury symptoms, such as mental health problems and posttraumatic stress disorder [4, 51–53]. Overall effect sizes of neuropsychological scores in mTBI patients are small, though deficits in cognitive performance include processing speed, learning, and memory [54, 55].

The detection rate of (acute) posttraumatic focal lesions in mTBI on 3T conventional MRI is much higher compared to head CT [56]. Conventional MRI within 2 weeks after mTBI is abnormal in 43% of patients [57]. In a cohort of patients with loss of consciousness and post-traumatic amnesia, conventional MRI detected parenchymal lesions in 75% of patients [56]. In 27–33% of pa-

tients with a normal admission head CT, early conventional brain MRI is abnormal [57, 58]. However, after 1 year, the prevalence of traumatic lesions has decreased considerably; only in 3% of cohorts of patients with post-concussion syndrome, MRI shows a structural abnormality, i.e., microhemorrhages, siderosis, or focal parenchymal defects [59].

In the majority of earlier studies using head CT and conventional 0.5–1.5T MRI sequences, cerebral microbleeds were not detected in mTBI patients. SWI is able to detect subtle brain abnormalities, especially microbleeds in mTBI. After introduction of SWI, a 1.5T study [41] showed 51 microbleeds in 16 mTBI patients. A 3T study [34] demonstrated 60 microbleeds in 26 patients and a prevalence of 23% microbleeds in mTBI patients [34, 41].

SWI studies revealed a positive relationship between the prevalence of traumatic microbleeds and injury severity. In minimal head trauma, no traumatic microbleeds are found [39]. On the other hand, microbleeds are not exclusive to moderate or severe TBI. In 27% of mTBI, in 47% of moderate, and in 58% of severe TBI patients, microbleeds are identified [23]. It is difficult to determine if traumatic microbleeds alone can predict cognitive outcome or persistent posttraumatic complaints. There is some evidence that mTBI patients with microbleeds may have higher symptom severity at 12 months and worse performance on tests of psychomotor speed and speed of information processing after 3 and 12 months [12, 34, 40]. More longitudinal studies are necessary to confirm the predictive value of SWI for posttraumatic complaints and cognitive outcome.

SWI alone is not helpful in determining the age of lesions, as both acute and chronic blood products demonstrate signal loss on T2 imaging. Another disadvantage of SWI is the indirect proof of axonal injury through microvascular shear injury [6]. Traumatic microbleeds should be recognized as a form of traumatic microvascular and not only axonal injury. Studies of mTBI combining SWI with diffusion tensor imaging may be helpful to provide further evidence of microstructural brain abnormalities underlying symptom chronification [19, 60]. SWI is a complementary, valuable imaging technique in mTBI, in patients with persistent posttraumatic complaints after 3 months.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

G.H. conceived the survey and drafted the manuscript; J.H. and J.N. contributed to the manuscript and approved the final version.

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